PHONOCARDIOGRAPHY

ITS APPLICATION TO CLINICAL MEDICINE*

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Introduction

Auscultation as a means of picking up heart sounds is mentioned by Hippocrates [1] and later, in 1628, by William Harvey,[2] but it was Laennec [3] in 1819 who first gave a precise description of the character of heart sounds. He evolved the wooden stethoscope. From then onwards interest in the mode of production of heart sounds was keen, and in 1835 the British Association for Advancement of Science [4] established a committee to elucidate the exact causes of heart sounds. In 1893 Hurthle [5] registered heart sounds graphically for the first time. He was followed a year later by Einthoven and Geluk,[6] and Frank [7] in 1904 evolved the segment capsule. Since then great strides have been made in perfecting registration of heart sounds and the causes of these sounds have been investigated experimentally and clinically with the aid of the phonocardiograph by various workers.[8-13]

Phonocardiography is the graphic registration of sounds produced in the heart and great vessels. Its chief advantage over auscultation is that:

1. Sounds can be recorded which are below the hearing threshold or inaudible owing to masking and fatigue effects on the ear from preceding loud sounds.
2. Sounds can be accurately timed in relation to the cardiac cycle.
3. It is possible to study the intensity, pitch and duration of sounds which have diagnostic significance.

By the use of microphones which transform sound vibrations into electric waves, it is possible with the aid of different types of bell and Bowles chest pieces to accentuate selected sounds and murmurs while others are attenuated. The stethoscopic microphone records sounds below the hearing threshold. It registers cardiac sounds and murmurs as they are produced in the chest wall. The logarithmic microphone registers sounds and murmurs which are normally audible, and when used with the Bowles type of chest piece can selectively accentuate high-pitched sounds and murmurs, and attenuate low-pitched ones. It is particularly useful in detecting high-pitched murmurs such as one encounters in aortic insufficiency.

Simultaneously with the heart sounds an electrocardiogram and/or a jugular phlebogram recording provide an adequate reference tracing to decide the circulatory events responsible for the sounds or murmurs. The phlebogram is particularly useful as it gives valuable information by portraying the different phases of cardiac activity. The *a* wave is due to auricular systole. The *c* wave is due to the onset of ventricular systole, and the *v* wave is due to the filling of the auricle, and its peak corresponds to the opening of the AV valves.

To evaluate the intensity of the sounds and murmurs a standard signal is provided in the machine, which has a sensitivity of 90 decibels above human hearing, and a frequency of 60 cycles per second. By comparing the two an approximate evaluation, in decibels, of the sounds and murmurs is obtained.

Over 1,500 phonocardiographic tracings have been recorded in the course of this study, and the tracings described here, are in general a reflection of the sounds and murmurs of the different cardiac conditions examined.

Two machines were used:

(1) The Cambridge Portable Electrocardiograph-Stethograph simple Trol model.

(2) The Sanborn Twin Beam Cardiette model 62.

GENESIS OF HEART SOUNDS

Heart sounds are produced by vibrations originating in the closure and opening of the valves, muscular contraction of the ventricles and the auricles, and the flow of blood through the large vessels.

Four heart sounds may be seen in the normal phonocardiogram and each may be broken down into several components.

In the *1st heart sound* 4 components are recognized. Its 1st component is represented by one or two low-frequency and low-amplitude vibrations due to 'development of tension in the ventricular muscle fibres which indicates the rise in intraventricular pressure'.[14] It is best demonstrated in the stethoscopic tracing at the apex of the heart beat. It commences at the peak of the R wave of the QRS complex of the electrocardiogram (ECG).

The 2nd and 3rd components—the valvular components—record the closure of the mitral and tricuspid valves and the opening of the semilunar valves. There is nearly always a clear separation between them. They are of high frequency and high amplitude. They end before the rise of the *c* wave of the jugular pulse, i.e. the beginning of the ejection phase.

The 4th component is considered to be due to the flow of blood in the large arteries during the phase of maximal ejection. It extends to the peak of the *c* wave of the jugular pulse.

The 2nd and 3rd components are high-frequency sounds, and are heard well with the ordinary stethoscope,

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but the 1st and 4th are of low-frequency and are seldom heard.

The total duration of the 1st heart sound varies considerably—from 0·1 to 0·16 seconds.

The *2nd heart sound* also consists of 4 components. The 1st component is a low-pitched vibration and is muscular in origin, due to ventricular relaxation in commencing diastole. The 2nd component is of high amplitude and high pitch, and represents the closure of the semilunar valves. The 3rd component is composed of one or two coarse vibrations of smaller amplitude and is vascular in origin, representing blood passing through arteries. The 4th component is represented by a low vibration, and is due to the opening of the mitral and tricuspid valves. It coincides with the apex of the *v* wave of the phlebogram, and becomes audible in mitral stenosis, when it is known as the opening snap of the mitral valve. Its duration varies from about 0·08 to 0·14 seconds.

The *3rd heart sound* occurs in diastole following closely on the 2nd sound and is commonly heard in young people up to the age of 20 years. It is uncommon after the 3rd decade. If it is heard after the age of 40 years a pathological condition of the heart should be suspected, e.g. hypertensive heart disease or cardiac infarction.[15] It coincides with the descending limb of the *v* wave of the phlebogram and is thought to be caused by vibrations of the ventricular wall during its distention by the inrush of blood from the auricle in the final phase of rapid ventricular filling. Sloan *et al.*[16] found that this sound was heard in 9·8% of university students between the ages of 17 and 32 years, but that it was recorded by the stethoscopic microphone in 100% and in only 39% when the logarithmic microphone was used. My stethoscopic tracings invariably show this sound in all young people. It is best heard at the apex and with the subject lying on the left side. It can be increased by pressure on the abdomen or raising the limbs on exercise. It occurs about 0·16 to 0·24 seconds after the beginning of the 2nd sound and has to be differentiated from other sounds in diastole, e.g. opening snap, splitting of the 2nd sound and auricular sounds. It is of low frequency, differing thus in quality from the opening snap, which is higher pitched and 0·03 to 0·14 (average about 0·07) seconds distant from the 2nd sound. It is not recorded in mitral stenosis, but if present in rheumatic valvular disease it is very suggestive of mitral incompetence.[17]

The *4th or auricular heart sound* is very seldom heard. This is due to the fact that it is of very low intensity and frequency and if present is often attenuated by a loud higher-pitched 1st heart sound. With the stethoscopic microphone it is recorded in 88% of normal cases, but only in 21% with the logarithmic microphone.[12] It is caused by muscular contraction of the auricle and the passage of blood through the valvular orifice. It is situated between the P and the Q waves of the ECG, and generally 0·08 to 0·14 seconds after the peak of the P wave (Figs. 1, 2 and 3).

Apart from the ordinary heart sounds other sounds are often present, which follow each other in rapid succession, and the ear is unable to appreciate their position in the cardiac cycle. The phonocardiograph becomes extremely important and offers the best way of differentiating such sounds.

Splitting of the 1st and 2nd Sounds

The 1st heart sound may be normally or abnormally split. There is usually a distinct separation between the 2nd and 3rd components, and this separation may become audible normally. When there is asynchronous closure of the mitral and tricuspid valves, as occurs in bundle-branch block, the splitting may be abnormally wide and may be confused with extra sounds (Fig. 4).

Splitting of the 2nd heart sound is normally found in healthy children in the pulmonary area and may be accentuated by deep inspiration. A split second sound is an indication of the existence of both aortic and pulmonary vessels, [18] and is of importance in congenital heart disease.[19] When present, the 1st component is due to closure of the aortic valves and the 2nd to the closure of the pulmonary valves. In congenital heart disease or pulmonary hypertension accentuation of one or other of the components of the 2nd heart sound is of diagnostic importance. In pulmonary hypertension the pulmonary element is louder than the aortic; in pulmonary stenosis on the other hand the aortic element is louder and the tracing comprises a very long single vibration. In the aortic and apical areas the 2nd sound is usually single.

An early single or double systolic sound following immediately on the 1st heart sound is sometimes heard in the precordial area in the 3rd or 4th interspace to the left of the sternum near the apex. It is short, high pitched and clicking in nature. It may be early, middle or late in systole. This sound was first described by Gallavardin[20] in 1913. He applied to it the terms 'pseudo-reduplication of the 1st heart sound' and 'telesystolic extracardiac sound.' He believed that the sound was produced by tugging on pleuro-pericardial adhesions during systole—a conclusion arrived at from necropsy findings in 3 patients who had exhibited the sound during life. It is called the 'mid-systolic click',[21] and also '*le claquement meso-systolique pleuro-pericardiale*'.[22] It may occur in association with spontaneous mediastinal emphysema and it may be produced by injecting air in the mediastinal tissues.[21] It has no cardiac pathological significance, and its importance lies in the fact that it is sometimes mistaken for evidence of heart disease (Fig. 5).

A similar click in early systole is often noted in the pulmonary area in conditions of pulmonary hypertension and dilatation of the pulmonary artery. It is called the 'semilunar opening click',[23] and is considered to be a valuable sign in the diagnosis of enlargement of the pulmonary artery with or without pulmonary hypertension.[24] This systolic click at the base of the heart, unlike the systolic click at the apex, is therefore of diagnostic importance (Fig. 6).

Variation in the Heart Sounds

The intensity of the 1st heart sound varies with certain conditions. Those who suffer from emphysema, obesity, pericardial effusion, myocardial disease or mitral regurgitation may have decreased 1st heart sounds. Increase in intensity of the 1st sound depends upon the vigour of the ventricular systole. Thus we get loud

sounds in exercise and emotional states, and in hyperthyroidism, anaemia, rheumatic fever and hypertension.

The conditions giving rise to change in intensity of the 1st heart sound has been discussed by various authors.[25, 8, 26, 23] Dean [26] in experiments with perfused cats' hearts showed that the septal cusps of the mitral valve underwent slight upward movement during the last moments of passive filling and then sharp downward opening movement with auricular contraction. Wolferth and Margolies [23] in a study of 500 cases noted that there is a relationship between the PR interval of the ECG and the intensity of the 1st heart sound. Thus with a PR interval of 0·04 to 0·08 seconds (normal range about 0·16 to 0·18 seconds) the 1st heart sound tended to be loud. Utilizing the above observations Levine [27] explains the variation of the 1st sound on the hypothesis that when the auricles contract the AV valves are driven down into the ventricles by the rush of blood, and when the ventricles fill the AV valves float upwards to higher levels in the ventricles. If the ventricles start to contract soon after the auricles, i.e. when the PR interval is short, the leaflets are still low in the ventricular cavity and are relaxed. They snap back a considerable distance and the sound of their closure is loud. When, however, the PR interval is long the leaflets have had time to float up higher in the ventricle, the slack in the margins of the leaflets is taken up and the distance to the orifice is short, then a faint sound is produced. This is well illustrated in complete heart block, where the changing intensity of the 1st heart sound is noted with the change in the PR interval and a clinical diagnosis can be made at the bedside on auscultation (Fig. 7).

Sounds and murmurs can be modified by varying the pressure of the chest piece against the chest wall. When an open bell is used, the skin bounded by the lip of the bell acts as a diaphragm and the fleshy portion under the skin acts as a damping medium. By heavy pressure, the skin under the bell is tightened, low-pitched sounds and murmurs are attenuated and high-pitched ones stand out more clearly and distinctly. Thus heavy pressure on the chest-piece will eliminate or reduce low-pitched sounds or murmurs and bring out more clearly high-pitched sounds or murmurs (e.g. a murmur of aortic incompetence). Low-pitched sounds and murmurs (e.g. 3rd heart sounds and gallop sounds) can best be demonstrated with light pressure (Fig. 8).

Triple and Quadruple Sounds

Extra sounds are encountered in systole and diastole. When in diastole and having a canter-like quality an extra sound is known as a gallop (Fig. 9). The extra sound in systole may be due to reduplication of the first heart sound or to a systolic click. When in diastole it may be due to reduplication to the 2nd heart sound, an opening snap of the mitral valve, a 3rd heart sound or an auricular sound. Quadruple sounds heard in the cardiac cycle may be due to a combination of sounds mentioned above.

A summation gallop is the name given to a sound due to merging of the 3rd and 4th heart sounds when the ventricular rate is rapid. It is low pitched and its importance lies in the fact that it may be mistaken for a mid-diastolic murmur of mitral stenosis (Fig. 10).

MURMURS

'Murmurs' are more difficult to record than 'sounds' because generally they are of low intensity and high frequency. In addition extraneous sounds are picked up such as respiratory sounds, friction between microphone and skin or hair of the chest wall, vibrations caused by tense muscles, or thoracic movement. It is most important, in order to have a clear record, to eliminate all extraneous sounds. A quiet room, accurate apposition of the chest-piece of the microphone, and perfect relaxation of the patient, are essential. The record should be taken with respiration completely suspended in full expiration or in the optimum position in the respiratory cycle.

Mechanism of Production of Murmurs

Corrigan [28] was the first to offer an explanation of murmurs. He attached a piece of intestine to a tap and found that when the tube was constricted a bruit and a thrill were produced beyond the constricted portion provided the rate of flow was sufficiently great. Two factors are therefore necessary for the production of murmurs: (1) variation in the calibre of the vessel, and (2) velocity of the blood flow.

Bondi [29] noted that in a tube with smooth walls a murmur could be produced only when the velocity of the flow exceeded 200 cm. per second. Below that critical velocity murmurs are produced only if some roughness or any other irregularity exists in the wall of the vessel, when eddies and turbulence in the stream are produced. If, however, the rate of flow is less than 100 cm. per second no murmur is produced even in tubes with rough linings and constrictions.

The cardiovascular system lends itself to the creation of murmurs. The heart has irregular walls, membranous structures, chordae tendiniae, papillary muscles and chambers of unequal size. Eddies and turbulence are therefore liable to be formed and murmurs produced, but before a murmur can occur the blood flow must attain a certain critical level of velocity. In a normal heart with a velocity less than 200 cm. per second no murmurs are produced. When, however, the velocity of the blood flow is increased beyond the critical level, e.g. in increase in pressure-gradients between chambers, in increase of heart rate during exercise, in hyperthyroidism etc., a murmur is produced. We can thus have functional and organic murmurs. The detection of a murmur, therefore, does not justify the conclusion that the heart is diseased and, contrariwise, the absence of a murmur does not necessarily mean that the heart is not diseased.

Murmurs appear during systole and diastole. It is well known that whereas diastolic murmurs generally denote some form of heart disease, systolic murmurs are frequently heard in healthy individuals without heart disease.

Systolic Murmurs

Levine [27] defines a systolic murmur as 'a sound, however faint, that continues well beyond the 1st heart sound and extends at least through the first quarter or third of systole'. The assessment of such a murmur

on a functional or organic basis is often extremely difficult. MacKenzie minimized the significance of systolic murmurs. Masters [30] advises that 'a loud systolic murmur at the apex should be considered organic until it can be proved otherwise'.

Freeman and Levine [31] classified systolic murmurs into 6 grades. Grade 1 is considered as the faintest murmur, which is generally not heard until one listens very carefully for a short while so as to get the ear tuned to its presence. Grade 2 is a slight murmur heard immediately the stethoscope is placed on the chest wall. Grade 3 is a moderate and grade 4 a loud murmur. Grade 5 is loud and harsh but the stethoscope must be on the chest wall for the murmur to be heard. Grade 6 is extremely loud, harsh and rasping and can be heard with the stethoscope just removed from the chest wall. Grades 1 and 2 are considered as functional and 3 to 6 as organic. Appreciation of different grades comes with auscultatory experience and a rough measure of the nature of the murmur, whether organic or innocent, can be made. In assessing a murmur, however, factors such as anaemia, hyperthyroidism, tachycardia, fever and hypertension should be ruled out before an organic basis for fainter murmurs is given.

Attempts have been made to establish the nature of a murmur by the phonocardiogram. Evans [32] assesses systolic murmurs as innocent when they occur in mid-systole or in late systole. Using limb lead 2 of the ECG as a reference tracing, he found that when a line is drawn from the S line to touch the simultaneous tracing of the phonocardiograph the innocent murmur commences a short distance after the S line but the organic murmur commences at the S line. This relationship, however, has been disproved by Cowen and Parnum,[33] who could find no clear-cut distinction between an innocent and an organic murmur on such relationship to the S line. My own tracings bear out the contentions of the latter authors. Harris and Freeman [34] have tried to distinguish innocent from organic systolic murmurs on the basis of 'low frequency vibrations and uniform wave pattern' (Fig. 11) as against a murmur of 'chaotic mixture of amplitudes and frequencies' (Fig. 12). While my tracings suggest this possibility, in a great number of instances it is quite impossible to assess the nature of the murmur on the phonocardiograph. A systolic murmur of grade 3 intensity and upwards is more likely to be organic. Such intensity is often manifested in the phonocardiograph by high amplitude of the vibration, commencing early in systole, and even following on the 1st heart sound and gradually decreasing in amplitude. However, it is important to remember that murmurs of slight intensity registered at one period in a heart which is considered to be perfectly healthy may at a later stage prove to be organic.

MITRAL STENOSIS

Durozier [35] in 1862 described the sound in mitral stenosis as 'ffout-tata-rou'. The 'ffout' corresponding to the presystolic crescendo murmur followed by a loud snapping 1st heart sound, 'tata' to the duplication of the 2nd heart sound, and 'rou' to the diastolic rumble. Rouches [36] in 1888 introduced the term '*claquement d'ouverture de la mitrale*' (the opening snap of the mitral valve) to explain the 2nd element of the 2nd heart sound. Since then studies made by various workers [23, 37, 39] have confirmed the accuracy of the above observations.

As a variety of acoustic phenomena occur in both systole and diastole the phonocardiogram is extremely important for an accurate differentiation of the various events. The 1st heart sound is generally extremely loud, snapping and delayed. Normally the maximal vibration of the 1st heart sound starts within 0·06 seconds of the onset of the QRS. In mitral stenosis the maximal vibration commences 0·07 seconds or more later.[38] Marked accentuation of the 1st heart sound is important 'as a reassuring sign that stenosis is a predominant lesion'.[40] This degree of intensity of the 1st heart sound can be explained on the basis of the position of the valves in the ventricular cavity at the moment of ventricular contraction. With stenosis the ventricles fill slowly; hence the cusps are low when the ventricles contract and the 1st sound is therefore loud (Fig. 13). In mitral incompetence and in the presence of calcified valves the 1st heart sound is usually soft. In incompetence the ventricles are filled rapidly and the valves are high, hence the sound is soft at the moment of closure. With calcification the valves hardly move; hence the sound is soft. In auricular fibrillation, when diastole is short the 1st heart sound is loud, when prolonged it is soft. A systolic murmur usually follows the 1st heart sound. Generally a loud pan-systolic murmur at the apex is associated with mitral incompetence, but in our experience the absence of such a murmur does not necessarily exclude incompetence (Fig. 14). Mounsey,[41] however, found that where an apical systolic murmur was absent on auscultation and on the phonocardiograph no incompetence was felt at mitral valvotomy.

The Opening Snap

This is now considered to be the most important indication of mitral stenosis. The sound is due to the sudden stretching of the stenosed valves with the inrush of blood from the left auricle.[42] It is high pitched and is usually heard in the mitral area inside the mid-clavicular line. It is also heard in the pulmonary area or along the left sternal border. The time interval between the beginning of the 2nd element of the 2nd heart sound and the opening snap varies from 0·03 to 0·14 seconds; the average is about 0·07 seconds. It is seen opposite the peak of the *v* wave of the phlebogram. It immediately precedes the mid-diastolic murmur, or there may be a small gap between it and the murmur. In cases where there is gross calcification of the mitral valves the opening snap may be absent. It is not abolished in auricular fibrillation, where its distance varies with the preceding RR interval. Where the preceding diastolic interval is increased the interval before the opening snap is greater than when the preceding interval is short.[41] The opening snap may be the earliest indication that mitral stenosis is in progress (Fig. 15). Levine and Harvey [43] relate how in 'some rheumatic cases observed over the course of years, at first only this opening snap could be heard without any detectable murmur in diastole. Then as time went on it became clear that a

slight and later a more pronounced diastolic rumble developed with this 3rd heart sound'.

The opening snap has to be differentiated from a split 2nd heart sound. The average interval between the beginning of the 2nd component of the 2nd heart sound and the beginning of the 3rd component is generally about 0·03 seconds or a little more in a split sound, whereas the opening snap averages about 0·07 seconds. It has also to be differentiated from a 3rd heart sound. The latter is lower pitched and its distance from the commencement of the 2nd heart sound is between 0·16 to 0·24 seconds. The peak of the *v* wave of the jugular pulse tracing is opposite the opening snap (Fig. 16), whereas the 3rd heart sound falls along the descending limb of the *v* wave (Fig. 2). It is rare to find a 3rd heart sound in mitral stenosis. We have often noted it in mitral incompetence.

The diastolic rumble generally starts after the opening snap but may be delayed and start after a short pause. It usually becomes more intense in the region of the 3rd heart sound owing to the more rapid flow of blood into the ventricle. It may fade in a diminuendo fashion and stop short of the 1st heart sound (Fig. 17), or it may continue in a crescendo manner to the 1st heart sound, giving rise to what is known as presystolic accentuation. It is of low frequency and is therefore often missed on auscultation. The bell chest-piece lightly applied will assist in its detection and the stethoscopic microphone is used for its registration. While a mid-diastolic murmur is generally considered to be an indication of mitral stenosis, Bland *et al.*[44] have noted that it may be present in a dilated ventricle and disappear as the heart returns to normal size. We have noted such diastolic murmurs disappear in cases which have been successfully operated for patent ductus arteriosus.

The Presystolic Murmur

When this murmur is noted at the apex of the heart it is considered as pathognomonic of mitral stenosis. It may be found alone or in association with a mid-diastolic murmur. It may continue in a crescendo manner to the 1st heart sound (Fig. 16), or there may actually be a slight pause between the mid-diastolic murmur and the presystolic murmur (Fig. 13). It may be the earliest sign of mitral stenosis. It is associated with auricular systole during which the velocity of the blood flow through the stenosed mitral valve is increased. When, however, the auricular contraction is feeble the velocity of the blood flow through the valve is diminished and the presystolic murmur becomes inaudible.

Mitral stenosis may be diagnosed erroneously on the basis of an acoustic impression of a presystolic murmur. The latter may be simulated by an intensely loud 1st heart sound, or by a duplicated 1st heart sound especially when the 2nd valvular component is louder than the first, thus giving the acoustic impression of a crescendo-like murmur (Fig. 18).

Valvotomy in well-selected cases, when successful, gives remarkable clinical improvement, but the auscultatory post-operative findings in the vast majority of cases still show the pre-operative sounds and murmurs, but reduced in intensity (Fig. 19). It is likely, however, that the persistence of the murmurs may be due to the the fact that the valves have not been split widely enough at operation. I am now informed that attempts are being made to split the valves wider. The results, assessment, contra-indications and limitations of valvotomy have been fully and ably discussed in this *Journal*.[45-48]

Tricuspid Stenosis and Incompetence

The murmurs in this condition are generally similar in nature and configuration to those encountered in mitral stenosis, but are heard best along the left and right sternal borders. The incompetent systolic murmur becomes more intense in full inspiration. The condition is usually associated with mitral stenosis (Fig. 20).

AORTIC STENOSIS AND REGURGITATION

The systolic murmur in aortic stenosis takes on a characteristic configuration. It is symmetrical, crescendo-decrescendo, starts late in systole, becomes intense in mid-systole, and ends before the 2nd heart sound. It takes on a diamond-shaped configuration. Its constancy is so invariable that it is extremely valuable as a diagnostic sign (Fig. 21). Occasionally it has to be differentiated from the systolic murmur of mitral incompetence in the mitral area. The diamond-shaped murmur recorded in the mitral area will give the answer (Fig. 22).

Basal Diastolic Murmurs

The murmurs of aortic and pulmonary regurgitation result from the backflow from the aortic and pulmonary vessels during diastole. They usually follow immediately on the 2nd heart sound, are blowing in character, high pitched and diminuendo throughout diastole. Wells *et al.*[49] have shown that the murmur may be crescendo-decrescendo in configuration and if the 2nd heart sound is single in configuration there may be a slight gap between the 2nd sound and the commencement of the murmur. When the 2nd sound is split there may be a few vibrations between the two components and the murmur therefore commences with the 1st component, thus giving the crescendo-decrescendo tracing. The early diastolic murmur is often very faint and is therefore frequently missed on auscultation. It is best recorded with the logarithmic microphone and with the Bowles chest piece (Fig. 23).

It is difficult to differentiate on the phonocardiograph between aortic incompetence and pulmonary incompetence. The clinical evidence of a collapsing pulse is of assistance. An early diastolic murmur which is best picked up at the lower end of the sternum is likely to be due to aortic incompetence. When the murmur is located in the pulmonary area it is more likely to be pulmonary incompetence.

A *musical diastolic murmur* due to aortic regurgitation is occasionally met with. Bellet *et al.*[50] have described such murmurs and have attributed them to retroversion of the aortic cusps which, in their opinion, is almost invariably associated with syphilis. The musical quality of the sound has suggested various descriptive terms, such as cooing dove, cuckoo clock, humming of a top or buzzing of a saw. The murmur has a frequency of 200 cycles per second. It is more intense and higher in pitch than a systolic murmur. It is very characteristic in

its configuration. It commences at the 2nd heart sound, which is indistinct and takes on a decrescendo character, fading as it approaches the 1st heart sound, which is hardly visible (Fig. 24).

A *musical systolic murmur* was encountered in a case which had recovered from subacute bacterial endocarditis. It is recorded in Fig. 25 and probably due to rupture of one of the mitral valves.

(A *musical extracardiac murmur* was heard in a case which presented itself as a problem regarding an extra sound in systole and a musical short late systolic murmur. The phonocardiograph shows a soft split 1st heart sound, a late systolic click and a very short late murmur the configuration of which shows a frequency typical of a musical murmur. Because of its association with the systolic click it was considered to be extracardiac in origin, probably pleuro-pericardial—Fig. 26).

CONGENITAL HEART DISEASE

The advent of cardiac surgery in recent years has changed the outlook of congenital heart disease completely. Phonocardiography plays an important part in assessing the nature of the lesion and assists greatly in the final diagnosis.

Patent Ductus Arteriosus. The murmur in this condition, likened in description to machinery, is continuous in character and is best heard in the pulmonary area. It may be heard at the back or along the left infraclavicular region. Starting late in systole it increases in intensity to the 2nd heart sound, which it envelops, and continues in a decrescendo manner throughout most or all diastole. It is most intense in systole (Fig. 27). A mid-diastolic murmur is sometimes noted in the mitral area (Fig. 28). It is suggested that this murmur is due to rapid ventricular filling of an enlarged left ventricle. It starts with the 3rd heart sound and is of lower frequency. After successful ligation the continuous murmur completely disappears, as does the mid-diastolic murmur. Sometimes a faint systolic murmur remains (Fig. 29).

Reversed Patent Ductus Arteriosus. A number of cases of patent ductus have been encountered which did not show the usual features of a continuous murmur. These cases were associated with cyanosis and invariably with pulmonary hypertension as proved by cardiac catheterization. The flow of blood instead of from the aorta to the pulmonary artery is shunted through the patent ductus in the reverse direction. The phonocardiograph shows the following features. The 1st and 2nd heart sounds are distinct and loud. The systolic murmur is early, short and decrescendo. A diastolic murmur follows on the 2nd heart sound, is mildly decrescendo and is prolonged, occupying practically the whole of diastole. Some of these cases were successfully ligated by Mr. D. Adler, and the murmurs, though still noticeable on the post-operative phonocardiogram, are reduced in intensity. Clinically the patients felt very much better (Fig. 30).

A patent ductus has to be differentiated from (1) arterio-venous fistula (Fig. 31), (2) venous hum (Fig. 32), and (3) the continuous murmur of ruptured sinus of Valsalva (Fig. 33).

The configuration of an arterio-venous fistula is similar to that of a patent ductus. It is usually very intense over the fistula and is transmitted over a wide area. Bone transmission is usually well marked, and, depending upon where the fistula is situated, the murmur can be heard at a distance in the skull, elbow etc. The mechanism of the murmur in these conditions follows the explanation of murmurs in general. There is an abrupt change in vessel diameter, an increase in the velocity of the blood flow, and an extreme gradient from a high-pressure level in the artery to the low pressure in the vein, giving rise to rapid flow through the fistula with consequent vibrations and eddies.

Pulmonary Stenosis. In pulmonary stenosis the murmur is registered best in the 2nd left interspace. The 1st sound is usually soft and the 2nd sound is single and represented by the aortic component. The systolic murmur is diamond-shaped, with vibrations of high frequency and high amplitude. It starts a short distance from the 1st heart sound, develops a crescendo configuration with maximum intensity beyond mid-systole, and reaches the 2nd heart sound in a decrescendo manner (Fig. 34). In some instances the systolic murmur after reaching the 2nd heart sound extends beyond it into diastole for a short distance. This early diastolic murmur is considered to be due either to post-stenotic dilatation of the pulmonary artery or to 'delay of termination of right ventricular systole, beyond the time of closure of the aortic component of the 2nd sound'[51] (Fig. 35).

In infundibular stenosis the murmur starts early in systole immediately after the 1st heart sound and ends a considerable distance before the 2nd sound. It is best located in the 4th left interspace (Fig. 36).

In mild pulmonary stenosis the 2nd sound is split, with the pulmonary component more accentuated than the aortic component. The systolic murmur is diamond-shaped and occupies practically the whole of systole (Fig. 37).

Ventricular Septal Defect. In this condition the systolic murmur is diamond-shaped and may spread over the whole of systole. The 2nd sound is split, the aortic component being of higher amplitude (Fig. 38).

Auricular Septal Defect. Here the 1st sound is broad and split and the 2nd sound is widely split, with the aortic component a little more intense, and is of high amplitude. The systolic murmur commences at the 1st heart sound and may stop short before the 2nd heart sound. A mid-diastolic murmur is often noted in the mitral area (Fig. 39).

Tetralogy of Fallot. The systolic murmur, when present, is found in the 2nd left interspace, is diamond-shaped, and is similar to the murmur noted in pulmonary stenosis (Fig. 40).

Coarctation of the Aorta. In this condition the 1st and 2nd heart sounds are usually well marked. The configuration of the systolic murmur depends on the site over which the microphone is placed. If it is over the stenosed area the murmur is diamond-shaped (Fig. 40). When the microphone is placed beyond the coarctation along the lower border of the sternum or at the back, the murmur is late in systole and is plateau-shaped (Fig. 42).

Sir James MacKenzie is reported to have said that the stethoscope should be thrown away because it does more harm than good. Since his time great strides have been made in cardiology and the value of auscultation has become fully established. The human ear, however, has inherent limitations. It is sensitive to vibrations whose frequencies lie between 20 and 20,000 cycles per second and, as the vibrations of heart sounds vary between 5 and 650 cycles per second, some cardiac vibrations become inaudible or are only heard by very keen and able observers. One can therefore appreciate the importance of phonocardiography, which registers sounds and murmurs as they are produced in the chest wall. Our cardiac clinic appreciated this important fact and a phonocardiographic department was established at its very inception. We have found phonocardiography extremely helpful in diagnosis of all sorts of cardiac conditions. It has given members of the staff confidence. It has been of great help in timing of heart sounds both normal and abnormal. It has taught us to appreciate split sounds, 3rd heart sounds, gallops, opening snaps. It has helped us to avoid pitfalls in diagnosis. It has proved of value where sounds are inaudible owing to masking and fatiguing effects on the ear from previous loud sounds. It has proved of inestimable value in mitral stenosis and it has saved many a patient who has been made a chronic cardiac invalid by erroneous auscultatory interpretation of sounds and murmurs. The pattern of vibrations of pulmonary and aortic stenosis is classic and is therefore very useful in the diagnosis of these conditions. It has proved of great value in congenital heart disease, where the configuration of murmurs and splitting of the 2nd heart sound are of diagnostic importance. The study of various patterns of tracings with systolic and diastolic vibrations has given us a better understanding of cardiology and its problems. Lastly, phonocardiography provides a permanent and lasting record of sounds and murmurs which by comparison at a later stage may prove to be of great prognostic value.

Phonocardiography has a great future. Not only does its usefulness lie in diagnostic assistance but it can and should be of value in teaching students to appreciate sounds and murmurs, for nothing is more valuable in clinical medicine than a visual appreciation of signs.

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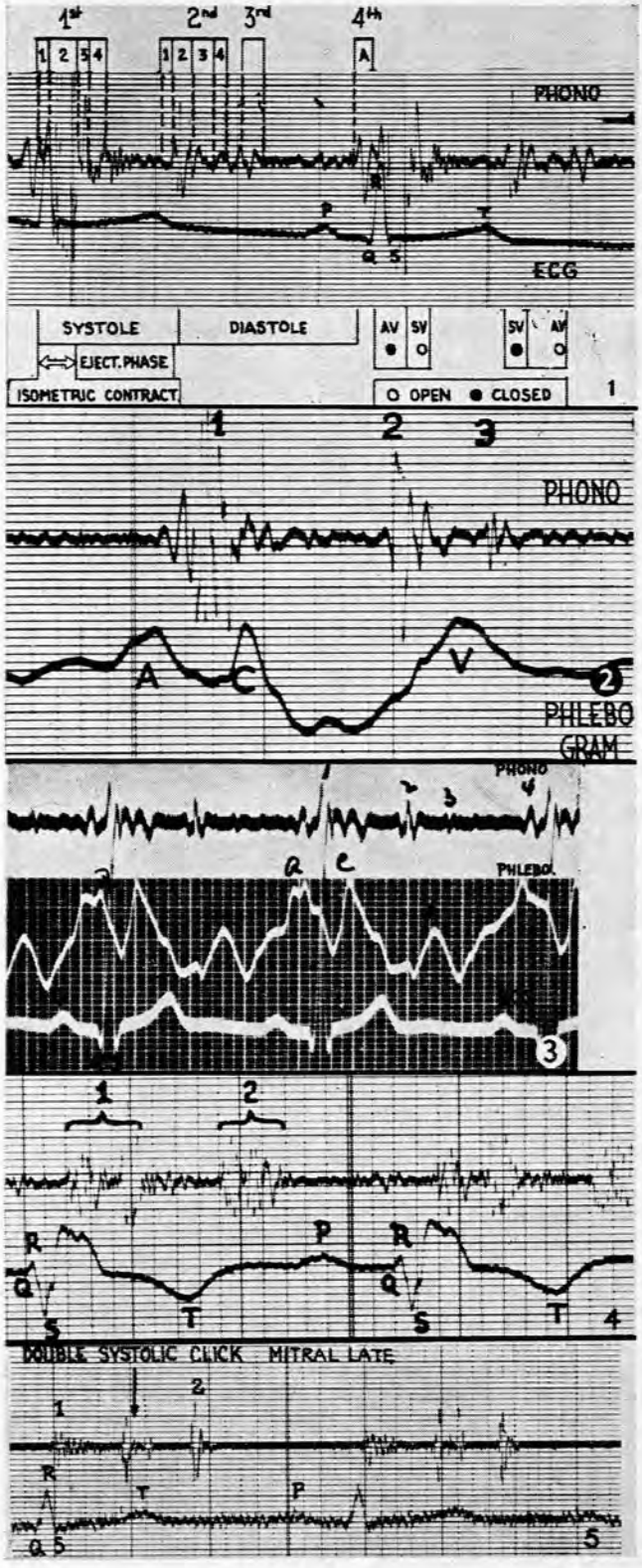
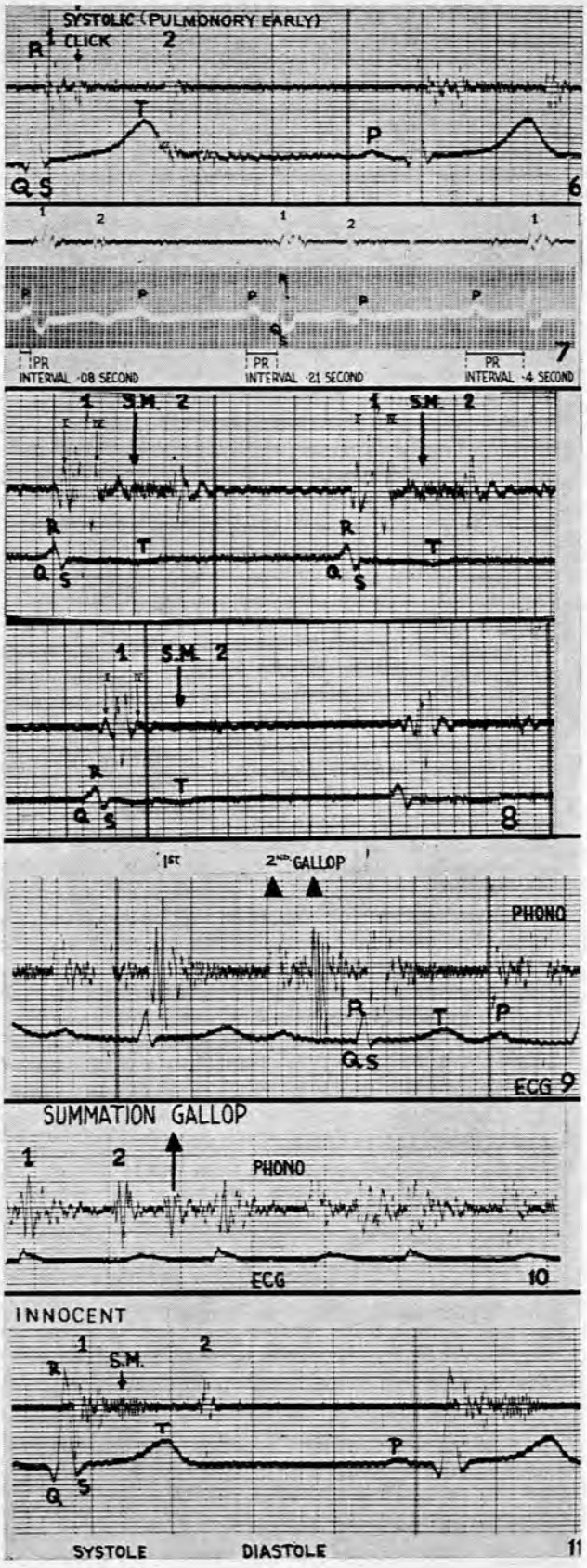
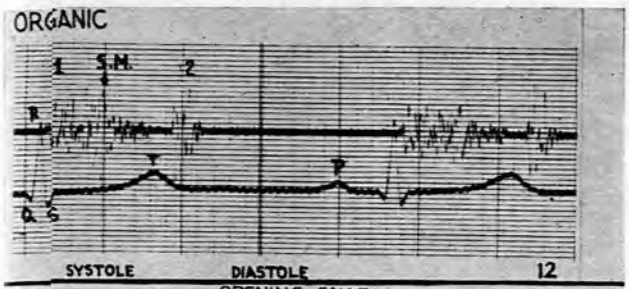
Fig. 1. Stethoscopic tracing. Normal phonocardiogram with simultaneous ECG showing the 4 heart sounds with the different components of the 1st and 2nd heart sounds. AV=auriculo-ventricular valves. SV=semilunar valves. A=auricular sound.
Fig. 2. Stethoscopic tracing. Normal phonocardiogram with simultaneous jugular phlebogram. Valvular component of the 1st sound ends before rise of c wave, apex of v wave opposite the 4th component of the 2nd heart sound and the 3rd heart sound opposite the descending limb of the v wave.
Fig. 3. Stethoscopic tracing. Simultaneous tracing of phonocardiogram, phlebogram and ECG.
Fig. 4. Stethoscopic tracing. Wide splitting of the 1st and 2nd heart sounds in right bundle-branch block.
Fig. 5. Logarithmic tracing. Late double systolic click in mitral area in a case of tumour of a lung.


1st
2nd
3rd
4th
1 2 3 4
1 2 3 4
A
PHONO
R
P
T
Q S
ECG
SYSTOLE
DIASTOLE
EJECT. PHASE
ISOMETRIC CONTRACT
AV
SV
SV
AV
O OPEN ● CLOSED
1
1 2 3
PHONO
A C V
2
PHLEBO GRAM
PHONO
a c
PHLEBO.
3
1 2
R P R
Q Q
S S T T
4
DOUBLE SYSTOLIC CLICK MITRAL LATE
1 2
R T P
Q S
5


SYSTOLIC (PULMONORY EARLY) CLICK
R 1 2
T
P
Q S
6
1 2 1 2 1
P P P P P
Q S
PR INTERVAL ·08 SECOND
PR INTERVAL ·21 SECOND
PR INTERVAL ·4 SECOND
7
1 S.M. 2
R T
Q S
1 S.M. 2
R T
Q S
1 S.M. 2
R T
Q S
8
1ST 2ND GALLOP
PHONO
R T P
Q S
ECG 9
SUMMATION GALLOP
1 2
PHONO
ECG 10
INNOCENT
1 2
S.M.
R T P
Q S
SYSTOLE DIASTOLE 11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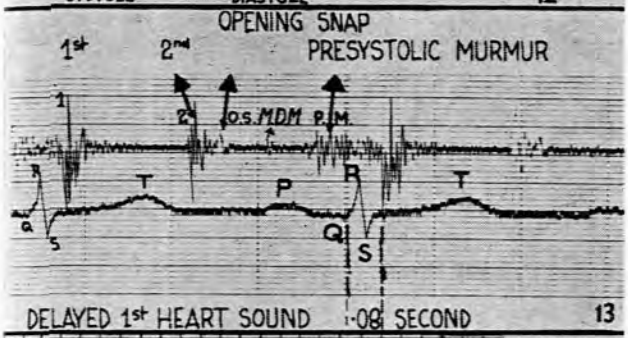
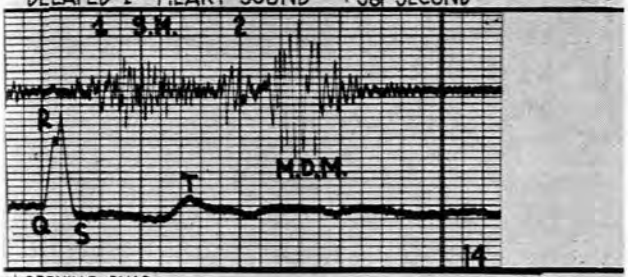
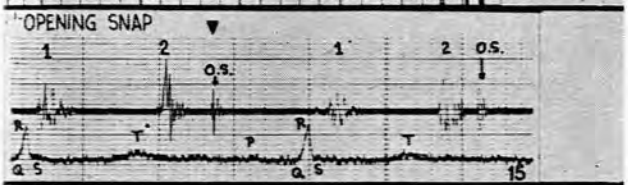
1
S.M.
2
R
T
P
Q
S
SYSTOLE
DIASTOLE
12


OPENING SNAP
1st
2nd
PRESYSTOLIC MURMUR
1
2
O.S. MDM P.M.
R
T
P
Q
S
DELAYED 1st HEART SOUND
·08 SECOND
13


1
S.M.
2
R
T
M.D.M.
Q
S
14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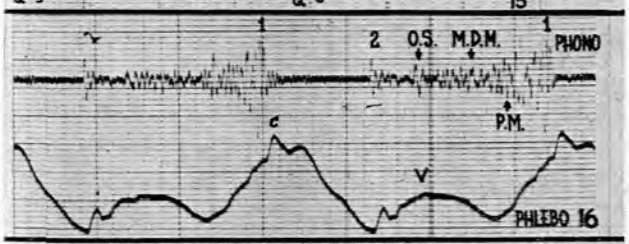
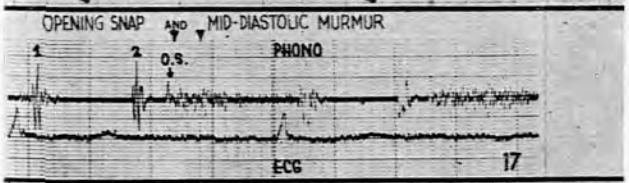
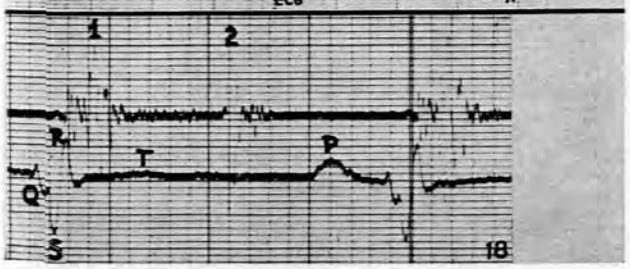
1
2
O.S.
1
2
O.S.
R
T
P
Q
S
15


1
2
O.S.
M.D.M.
1
PHONO
P.M.
c
v
PHLEBO 16


OPENING SNAP
AND
MID-DIASTOLIC MURMUR
1
2
O.S.
PHONO
ECG
17


1
2
R
T
P
Q
S
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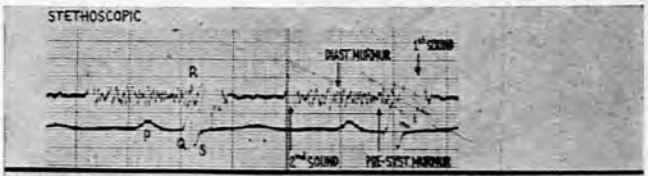
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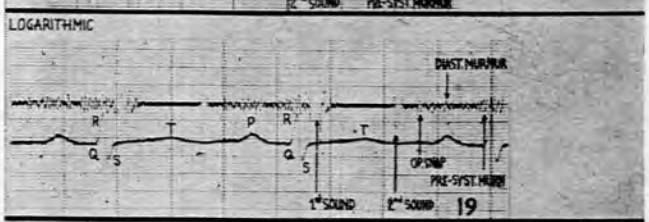
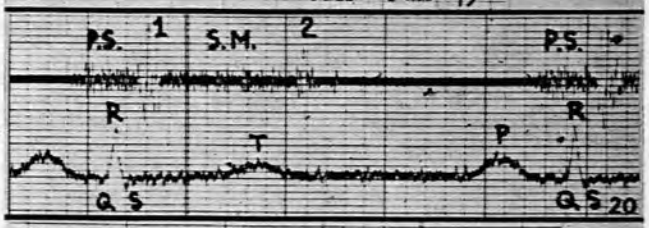
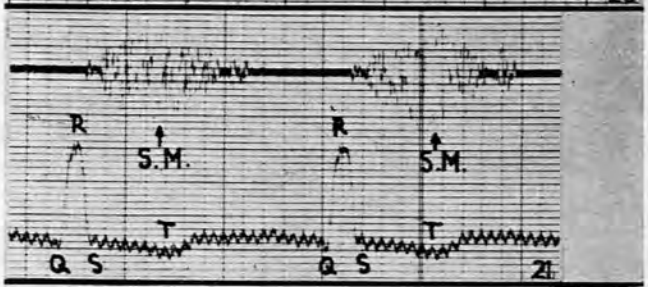
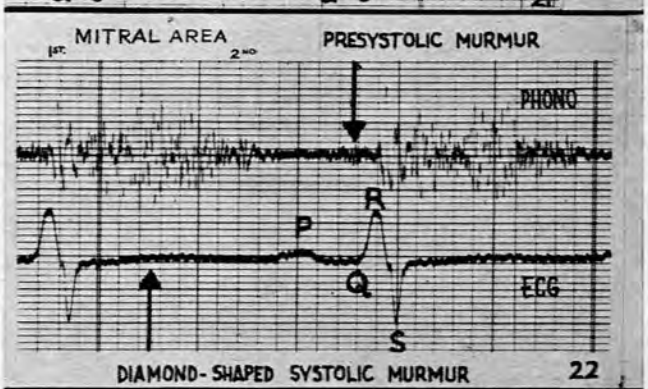
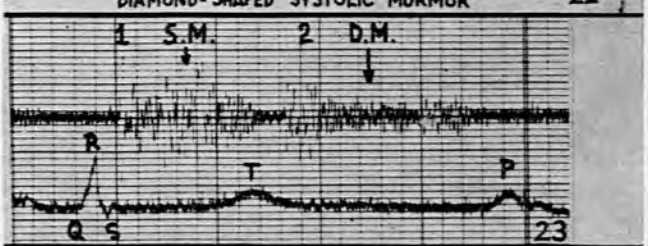
DIAST. MURMUR
1st SOUND
R
P
Q
S
2nd SOUND
PRE-SYST. MURMUR

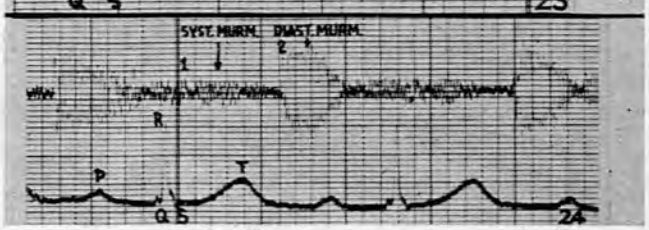

LOGARITHMIC
DIAST. MURMUR
R
T
P
Q
S
OP. SNAP
PRE-SYST. MURM.
1st SOUND
1st SOUND
19


P.S.
1
S.M.
2
P.S.
R
T
P
R
Q
S
Q
S
20


R
S.M.
R
S.M.
T
T
Q
S
Q
S
21


MITRAL AREA
1st
2nd
PRESYSTOLIC MURMUR
PHONO
R
P
Q
ECG
S
DIAMOND-SHAPED SYSTOLIC MURMUR
22


1
S.M.
2
D.M.
R
T
P
Q
S
23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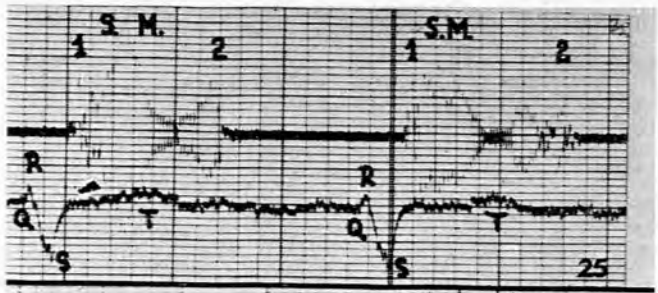
DIAST. MURM.
1
2
R
P
T
Q
S
24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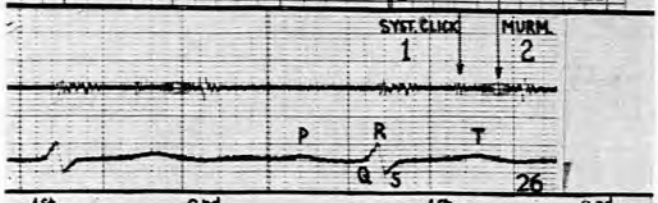
S.M.
25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MURM.
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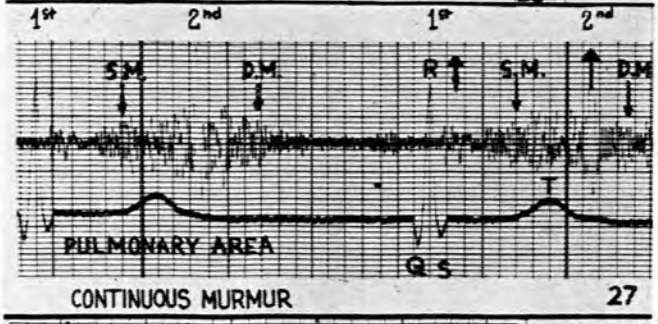
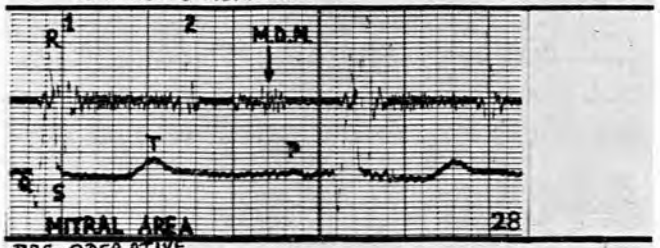

1st
2nd
1st
2nd
S.M.
D.M.
S.M.
D.M.
PULMONARY AREA
CONTINUOUS MURMUR
27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MITRAL AREA
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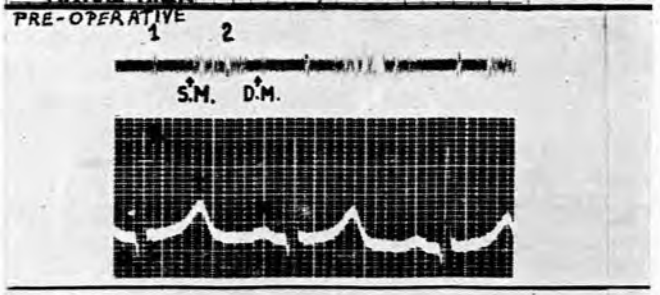
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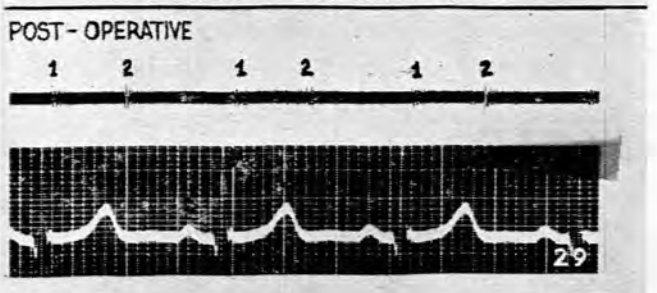
D.M.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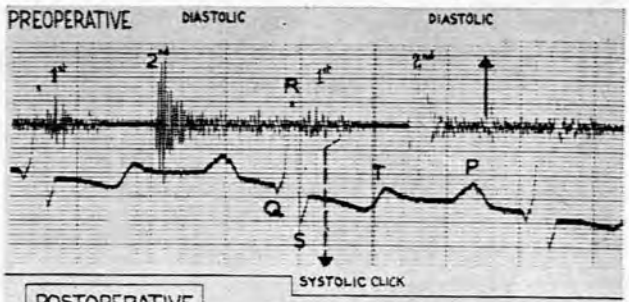
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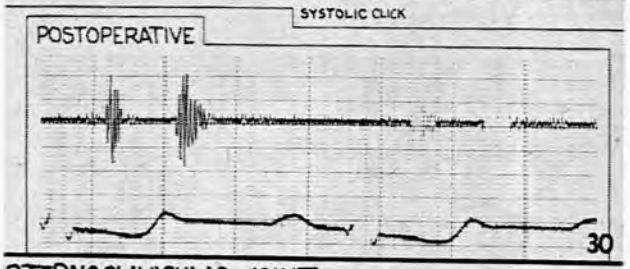
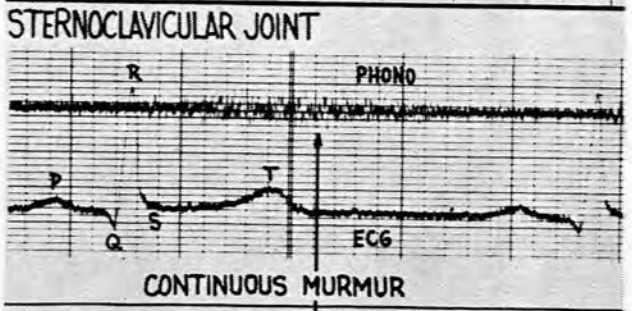
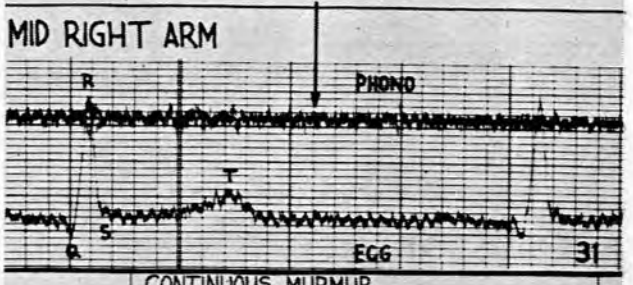
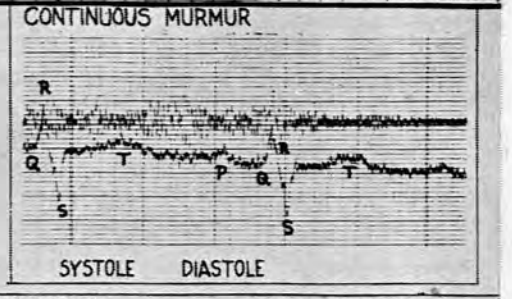
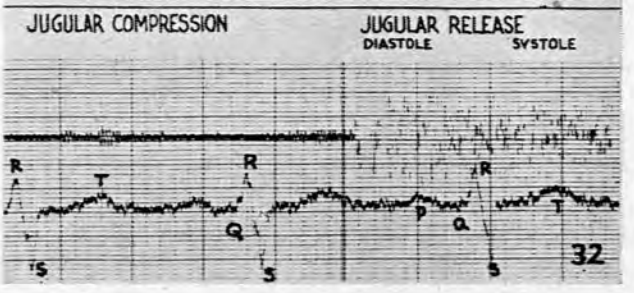
DIASTOLIC
SYSTOLIC CLICK

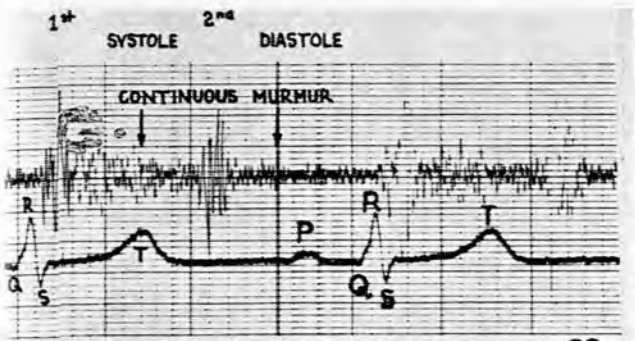

POSTOPERATIVE
30


STERNOCLAVICULAR JOINT
PHONO
ECG
CONTINUOUS MURMUR

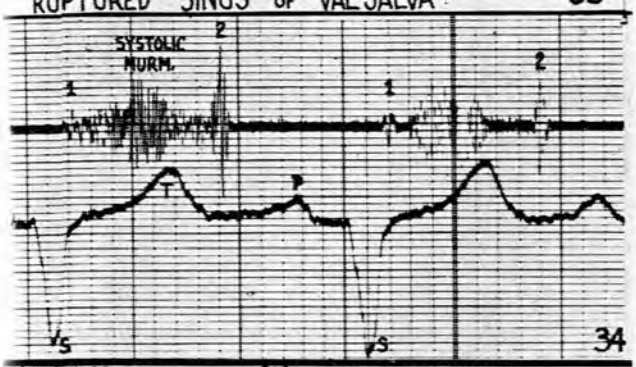

MID RIGHT ARM
PHONO
ECG
31


CONTINUOUS MURMUR
SYSTOLE
DIASTOLE

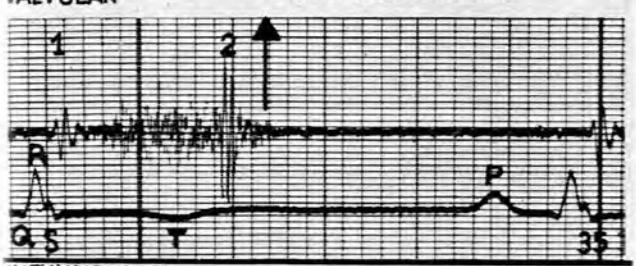

JUGULAR COMPRESSION
JUGULAR RELEASE
DIASTOLE
SYSTOLE
32



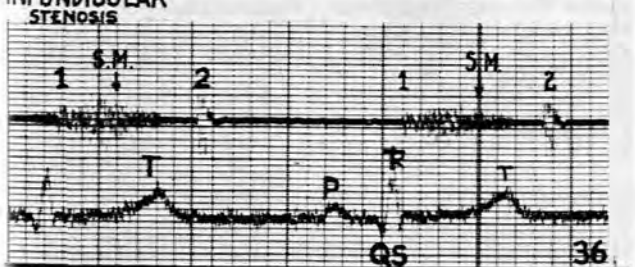
RUPTURED SINUS OF VALSALVA 33



VALVULAR EARLY DIASTOLIC MURMUR

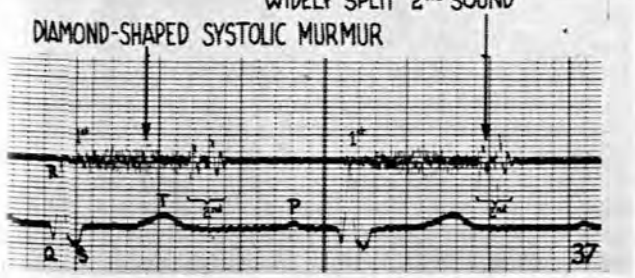


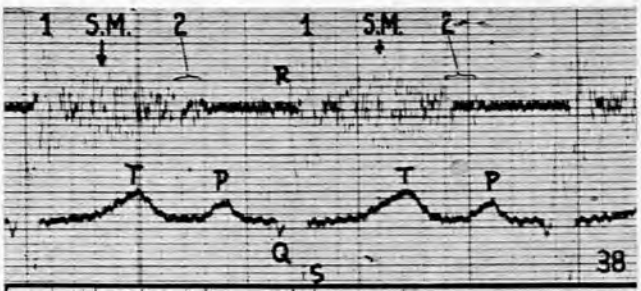
INFUNDIBULAR STENOSIS

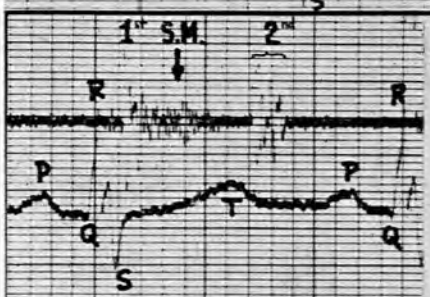


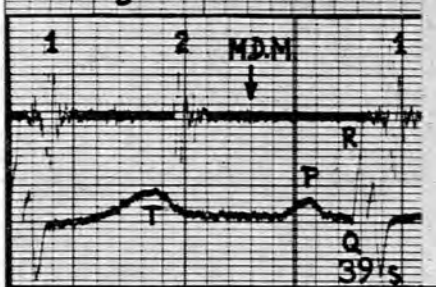
WIDELY SPLIT 2nd SOUND

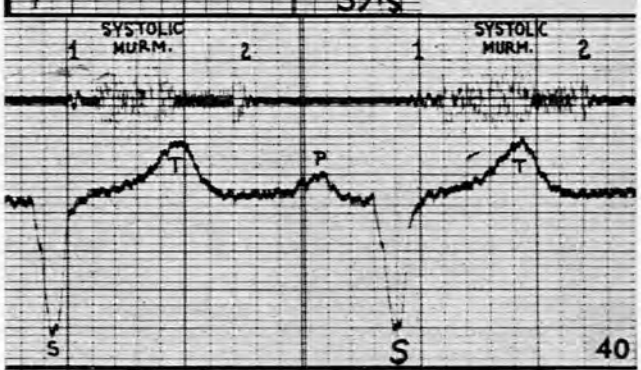
DIAMOND-SHAPED SYSTOLIC MURMUR



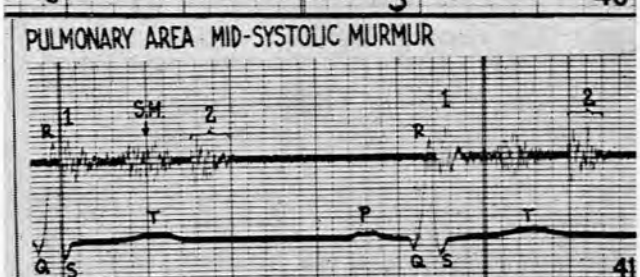








PULMONARY AREA MID-SYSTOLIC MURMUR



STERNAL AREA LATE SYSTOLIC MURMUR

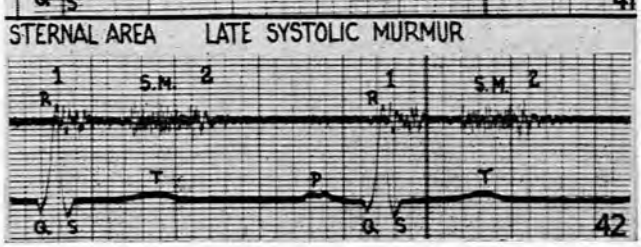


Fig. 6. Logarithmic tracing. Early systolic click in pulmonary area in a case of pulmonary dilatation and hypertension.
Fig. 7. Stethoscopic tracing. Changing intensity of the 1st heart sound in complete heart block. With P.R. interval of 0·08 seconds the duration and amplitude are 0·13 seconds and 11 mm.; with P.R. interval of 0·21 seconds the duration and amplitude are 0·08 seconds and 9·5 mm.; and with an interval of 0·4 seconds the duration and amplitude are 0·1 seconds and 8·5 mm.
Fig. 8. Changing sounds and murmurs with varying pressure. Upper tracing, light pressure. The 1st heart sound is broad and loud with 1st and 4th low-intensity components high in amplitude and the systolic murmur showing low-frequency and high-frequency vibrations. In the lower tracing with heavy pressure the 1st and 4th components of the 1st heart sound are attenuated, and only high-frequency and low-amplitude vibrations are noted in systole.
Fig. 9. Gallop rhythm. The extra sound in diastole had a canter-like quality.
Fig. 10. Summation gallop. The sound in diastole gave the impression of a rumbling diastolic murmur. The tracing shows summation of 3rd and 4th heart sounds.
Fig. 11. Innocent systolic murmur. Note low-frequency vibrations and uniform pattern.
Fig. 12. Organic systolic murmur. Note vibrations of irregular mixture of amplitudes and frequencies.
Fig. 13. Mitral stenosis. Note delayed 1st heart sounds, opening snap, presystolic murmur and faint low-grade diastolic murmur (M.D.M.). Note slight pause between diastolic and presystolic murmurs.
Fig. 14. Mitral incompetence and mitral stenosis. Note loud pan-systolic murmur, soft 1st heart sound and loud mid-diastolic murmur (M.D.M.).
Fig. 15. Mitral stenosis. Note opening snap 0·13 seconds from commencement of valvular component of 2nd sound. It was the only indication of mitral stenosis. The 1st heart sound delayed 0·08 seconds.
Fig. 16. Mitral stenosis. Opening snap (O.S.) opposite the v wave of the phlebogram, followed by a mid-diastolic murmur (M.D.M.) and with presystolic accentuation (P.M.). Note slight pause between opening snap and murmur.
Fig. 17. Mitral stenosis. Note delayed 1st heart sound 0·07 seconds, opening snap (O.S.) and mid-diastolic murmur fading towards 1st heart sound.
Fig. 18. Split 1st and 2nd heart sounds gave acoustic impression of mitral stenosis. The 2nd valvular component of the 1st sound is louder and gave the impression of a crescendo presystolic murmur and the 2nd valvular component of the 2nd heart sound gave the impression of an opening snap.
Fig. 19. Mitral stenosis. Post-valvotomy. Note presence of murmurs including opening snap (O.S.) in logarithmic tracing.
Fig. 20. Tricuspid stenosis. Note pan-systolic murmur (S.M.) and presystolic murmur (P.S.).
Fig. 21. Aortic stenosis. Note very soft 1st heart sound and diamond-shaped configuration of the systolic murmur (S.M.).
Fig. 22. Aortic stenosis and mitral stenosis. Mitral area. Note diamond-shaped configuration of the systolic murmur (S.M.) and the presystolic murmur (P.S.) and loud 1st heart sound of mitral stenosis.
Fig. 23. Aortic stenosis and aortic incompetence. Aortic area. Note diamond-shaped systolic murmur (S.M.) and early prolonged diastolic murmur (D.M.).
Fig. 24. Musical diastolic murmur (cooing dove). Note configuration and high frequency and high amplitude.
Fig. 25. Musical systolic murmur (S.M.) following subacute bacterial endocarditis.
Fig. 26. Extra-cardic musical murmur and systolic click.
Fig. 27. Patent ductus arteriosus. Pulmonary area. Note typical continuous murmur. Systolic louder than the diastolic murmur.
Fig. 28. Patent ductus. Mitral area. Systolic murmur only. Note mid-diastolic murmur (M.D.M.).
Fig. 29. Patent ductus. Upper tracing pre-operative; note continuous murmur. Lower tracing post-operative; note disappearance of murmur.
Fig. 30. Reversed patent ductus, proved by catheterization and operation. Upper tracing; note systolic click (indication of pulmonary hypertension), soft systolic murmur and loud 2nd heart sound with decrescendo diastolic murmur. Lower tracing post-operative; note disappearance of murmur.
Fig. 31. Arterio-venous fistula. Upper tracing, continuous murmur. Lower tracing, radiation of murmur along bone to elbow.
Fig. 32. Venous hum. Upper tracing, continuous murmur, diastolic louder than systolic. Lower tracing with jugular compression, note disappearance of murmur and reappearance with release of jugular vein.
Fig. 33. Ruptured sinus of Valsalva. Note continuous murmur.
Fig. 34. Pulmonary stenosis. Note diamond-shaped systolic murmur and single 2nd heart sound.
Fig. 35. Pulmonary stenosis, valvular type. Note systolic murmur reaches 2nd heart sound. Early diastolic murmur.
Fig. 36. Pulmonary stenosis, infundibular type. Note early diamond-shaped systolic murmur and single 2nd heart sound.
Fig. 37. Pulmonary stenosis, mild degree. Note diamond-shaped systolic murmur and widely split 2nd heart sound.
Fig. 38. Ventricular septal defect proved by catheterization. Note diamond-shaped systolic murmur and widely split 2nd heart sound.
Fig. 39. Auricular septal defect, proved by catheterization. Upper tracing, pulmonary area; note early systolic murmur and split 2nd heart sound. Lower tracing, mitral area; note mid-diastolic murmur (M.D.M.).
Fig. 40. Tetralogy of Fallot. Pulmonary area. Note diamond-shaped systolic murmur.
Fig. 41. Coarctation of aorta. Over coarctation murmur is diamond-shaped. Split 2nd sound.
Fig. 42. Coarctation of aorta. Lower border of sternum. Murmur late (S.M.).